Douglas R Higgs

List of Publications by Year
in descending order

155 papers	15,300 citations	14655 66 h-index	19749 117 g-index
167 all docs	167 docs citations	167 times ranked	14287 citing authors

#	Article	IF	Citations
1	Scalable in vitro production of defined mouse erythroblasts. PLoS ONE, 2022, 17, e0261950.	2.5	8
2	The chromatin remodeller ATRX facilitates diverse nuclear processes, in a stochastic manner, in both heterochromatin and euchromatin. Nature Communications, 2022, 13, .	12.8	20
3	Genetic and functional insights into CDA-I prevalence and pathogenesis. Journal of Medical Genetics, 2021, 58, 185-195.	3.2	9
4	The relationship between genome structure and function. Nature Reviews Genetics, 2021, 22, 154-168.	16.3	160
5	The mouse alpha-globin cluster: a paradigm for studying genome regulation and organization. Current Opinion in Genetics and Development, 2021, 67, 18-24.	3.3	21
6	A remarkable case of HbH disease illustrates the relative contributions of the Î±-globin enhancers to gene expression. Blood, 2021, 137, 572-575.	1.4	6
7	Enhancers predominantly regulate gene expression during differentiation via transcription initiation. Molecular Cell, 2021, 81, 983-997.e7.	9.7	27
8	A gain-of-function single nucleotide variant creates a new promoter which acts as an orientation-dependent enhancer-blocker. Nature Communications, 2021, 12, 3806.	12.8	18
9	Reactivation of a developmentally silenced embryonic globin gene. Nature Communications, 2021, 12, 4439.	12.8	19
10	Testing the super-enhancer concept. Nature Reviews Genetics, 2021, 22, 749-755.	16.3	53
11	Recapitulation of erythropoiesis in congenital dyserythropoietic anemia type I (CDA-I) identifies defects in differentiation and nucleolar abnormalities. Haematologica, 2021, 106, 2960-2970.	3.5	10
12	Systematic integration of GATA transcription factors and epigenomes via IDEAS paints the regulatory landscape of hematopoietic cells. IUBMB Life, 2020, 72, 27-38.	3.4	8
13	Loss of Extreme Long-Range Enhancers in Human Neural Crest Drives a Craniofacial Disorder. Cell Stem Cell, 2020, 27, 765-783.e14.	11.1	101
14	Dynamics of the 4D genome during in vivo lineage specification and differentiation. Nature Communications, 2020, 11, 2722.	12.8	79
15	An evolutionarily ancient mechanism for regulation of hemoglobin expression in vertebrate red cells. Blood, 2020, 136, 269-278.	1.4	16
16	An integrative view of the regulatory and transcriptional landscapes in mouse hematopoiesis. Genome Research, 2020, 30, 472-484.	5.5	38
17	A Dynamic Folded Hairpin Conformation Is Associated with Î±-Globin Activation in Erythroid Cells. Cell Reports, 2020, 30, 2125-2135.e5.	6.4	38
18	ATR-16 syndrome: mechanisms linking monosomy to phenotype. Journal of Medical Genetics, 2020, 57, 414-421.	3.2	7

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19	The bipartite TAD organization of the X-inactivation center ensures opposing developmental regulation of Tsix and Xist. Nature Genetics, 2019, 51, 1024-1034.	21.4	60
20	A revised model for promoter competition based on multi-way chromatin interactions at the Î±-globin locus. Nature Communications, 2019, 10, 5412.	12.8	60
21	Molecular Basis and Genetic Modifiers of Thalassemia. Hematology/Oncology Clinics of North America, 2018, 32, 177-191.	2.2	93
22	A tissue-specific self-interacting chromatin domain forms independently of enhancer-promoter interactions. Nature Communications, 2018, 9, 3849.	12.8	62
23	Single-allele chromatin interactions identify regulatory hubs in dynamic compartmentalized domains. Nature Genetics, 2018, 50, 1744-1751.	21.4	150
24	Robust CRISPR/Cas9 Genome Editing of the HUDEP-2 Erythroid Precursor Line Using Plasmids and Single-Stranded Oligonucleotide Donors. Methods and Protocols, 2018, 1, 28.	2.0	17
25	Potential new approaches to the management of the Hb Bartâ€™s hydrops fetalis syndrome: the most severe form of Î±-thalassemia. Hematology American Society of Hematology Education Program, 2018, 2018, 353-360.	2.5	25
26	How to Tackle Challenging ChIP-Seq, with Long-Range Cross-Linking, Using ATRX as an Example. Methods in Molecular Biology, 2018, 1832, 105-130.	0.9	7
27	How best to identify chromosomal interactions: a comparison of approaches. Nature Methods, 2017, 14, 125-134.	19.0	124
28	Selective silencing of Î±-globin by the histone demethylase inhibitor IOX1: a potentially new pathway for treatment of Î²-thalassemia. Haematologica, 2017, 102, e80-e84.	3.5	33
29	Functional characterisation of cis-regulatory elements governing dynamic Eomes expression in the early mouse embryo. Development (Cambridge), 2017, 144, 1249-1260.	2.5	32
30	An international registry of survivors with Hb Bart's hydrops fetalis syndrome. Blood, 2017, 129, 1251-1259.	1.4	59
31	The chromatin remodelling factor <scp>ATRX</scp> suppresses Râ€loops in transcribed telomeric repeats. EMBO Reports, 2017, 18, 914-928.	4.5	99
32	DNA methylation of intragenic CpG islands depends on their transcriptional activity during differentiation and disease. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, E7526-E7535.	7.1	125
33	Editing an Î±-globin enhancer in primary human hematopoietic stem cells as a treatment for Î²-thalassemia. Nature Communications, 2017, 8, 424.	12.8	85
34	Tissue-specific CTCFâ€“cohesin-mediated chromatin architecture delimits enhancer interactions and function in vivo. Nature Cell Biology, 2017, 19, 952-961.	10.3	179
35	Between form and function: the complexity of genome folding. Human Molecular Genetics, 2017, 26, R208-R215.	2.9	20
36	Robust detection of chromosomal interactions from small numbers of cells using low-input Capture-C. Nucleic Acids Research, 2017, 45, e184-e184.	14.5	27

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37	Understanding Î±â€globin gene regulation and implications for the treatment of Î²â€thalassemia. Annals of the New York Academy of Sciences, 2016, 1368, 16-24.	3.8	44
38	Genetic dissection of the Î±-globin super-enhancer in vivo. Nature Genetics, 2016, 48, 895-903.	21.4	308
39	MicroRNAs of the miR-290â€“295 Family Maintain Bivalency in Mouse Embryonic Stem Cells. Stem Cell Reports, 2016, 6, 635-642.	4.8	24
40	KrÃ¼ppeling erythropoiesis: an unexpected broad spectrum of human red blood cell disorders due to KLF1 variants. Blood, 2016, 127, 1856-1862.	1.4	124
41	Transcription factors LRF and BCL11A independently repress expression of fetal hemoglobin. Science, 2016, 351, 285-289.	12.6	260
42	Multiplexed analysis of chromosome conformation at vastly improved sensitivity. Nature Methods, 2016, 13, 74-80.	19.0	225
43	Suppression of the alternative lengthening of telomere pathway by the chromatin remodelling factor ATRX. Nature Communications, 2015, 6, 7538.	12.8	219
44	ATRX Plays a Key Role in Maintaining Silencing at Interstitial Heterochromatic Loci and Imprinted Genes. Cell Reports, 2015, 11, 405-418.	6.4	152
45	Î±-Globin as a molecular target in the treatment of Î²-thalassemia. Blood, 2015, 125, 3694-3701.	1.4	102
46	An international effort to cure a global health problem: A report on the 19th Hemoglobin Switching Conference. Experimental Hematology, 2015, 43, 821-837.	0.4	7
47	ATRX Dysfunction Induces Replication Defects in Primary Mouse Cells. PLoS ONE, 2014, 9, e92915.	2.5	84
48	Mutations in KrÃ¼ppel-like factor 1 cause transfusion-dependent hemolytic anemia and persistence of embryonic globin gene expression. Blood, 2014, 123, 1586-1595.	1.4	76
49	Analysis of hundreds of cis-regulatory landscapes at high resolution in a single, high-throughput experiment. Nature Genetics, 2014, 46, 205-212.	21.4	417
50	Differential regulation of the Î±-globin locus by KrÃ¼ppel-like factor 3 in erythroid and non-erythroid cells. BMC Molecular Biology, 2014, 15, 8.	3.0	11
51	The chromatin remodeller ATRX: a repeat offender in human disease. Trends in Biochemical Sciences, 2013, 38, 461-466.	7.5	103
52	Analysis of Sequence Variation Underlying Tissue-specific Transcription Factor Binding and Gene Expression. Human Mutation, 2013, 34, 1140-1148.	2.5	10
53	Causes and Consequences of Chromatin Variation between Inbred Mice. PLoS Genetics, 2013, 9, e1003570.	3.5	18
54	High-resolution analysis of cis -acting regulatory networks at the Î±-globin locus. Philosophical Transactions of the Royal Society B: Biological Sciences, 2013, 368, 20120361.	4.0	12

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55	Homozygous mutations in a predicted endonuclease are a novel cause of congenital dyserythropoietic anemia type I. Haematologica, 2013, 98, 1383-1387.	3.5	71
56	The Molecular Basis of Â-Thalassemia. Cold Spring Harbor Perspectives in Medicine, 2013, 3, a011718-a011718.	6.2	106
57	An interspecies analysis reveals a key role for unmethylated CpG dinucleotides in vertebrate Polycomb complex recruitment. EMBO Journal, 2012, 31, 317-329.	7.8	173
58	Thalassaemia. Lancet, The, 2012, 379, 373-383.	13.7	371
59	RNA discrimination. Nature, 2012, 482, 310-311.	27.8	104
60	Intragenic Enhancers Act as Alternative Promoters. Molecular Cell, 2012, 45, 447-458.	9.7	237
61	Nprl3 is required for normal development of the cardiovascular system. Mammalian Genome, 2012, 23, 404-415.	2.2	38
62	Combinatorial readout of histone H3 modifications specifies localization of ATRX to heterochromatin. Nature Structural and Molecular Biology, 2011, 18, 777-782.	8.2	187
63	Polycomb eviction as a new distant enhancer function. Genes and Development, 2011, 25, 1583-1588.	5.9	78
64	Global gene expression analysis of human erythroid progenitors. Blood, 2011, 117, e96-e108.	1.4	95
65	Codanin-1 mutations in congenital dyserythropoietic anemia type 1 affect HP1Î± localization in erythroblasts. Blood, 2011, 117, 6928-6938.	1.4	58
66	Systematic documentation and analysis of human genetic variation in hemoglobinopathies using the microattribution approach. Nature Genetics, 2011, 43, 295-301.	21.4	142
67	Generation of bivalent chromatin domains during cell fate decisions. Epigenetics and Chromatin, 2011, 4, 9.	3.9	54
68	Functional significance of mutations in the Snf2 domain of ATRX. Human Molecular Genetics, 2011, 20, 2603-2610.	2.9	46
69	ATRX: Taming tandem repeats. Cell Cycle, 2010, 9, 4605-4606.	2.6	4
70	Î±-thalassaemia. Orphanet Journal of Rare Diseases, 2010, 5, 13.	2.7	417
71	Distinct Factors Control Histone Variant H3.3 Localization at Specific Genomic Regions. Cell, 2010, 140, 678-691.	28.9	1,069
72	ATR-X Syndrome Protein Targets Tandem Repeats and Influences Allele-Specific Expression in a Size-Dependent Manner. Cell, 2010, 143, 367-378.	28.9	365

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73	The Molecular Basis of Î±-Thalassemia: A Model for Understanding Human Molecular Genetics. Hematology/Oncology Clinics of North America, 2010, 24, 1033-1054.	2.2	36
74	The Molecular Basis of Î± Thalassemia. , 2009, , 241-265.		1
75	Nuclear Factors That Regulate Erythropoiesis. , 2009, , 62-85.		3
76	THE MOLECULAR, CELLULAR, AND GENETIC BASIS OF HEMOGLOBIN DISORDERS. , 2009, , 1-2.		0
77	The Pathophysiology and Clinical Features of Î± Thalassaemia. , 2009, , 266-295.		7
78	Adventitious changes in long-range gene expression caused by polymorphic structural variation and promoter competition. Proceedings of the National Academy of Sciences of the United States of America, 2009, 106, 21771-21776.	7.1	77
79	Research Highlights. Epigenomics, 2009, 1, 231-234.	2.1	0
80	The role of Xâ€inactivation in the gender bias of patients with acquired Î±â€thalassaemia and myelodysplastic syndrome (ATMDS). British Journal of Haematology, 2009, 144, 538-545.	2.5	9
81	Chromosome looping at the human Î±-globin locus is mediated via the major upstream regulatory element (HS â^'40). Blood, 2009, 114, 4253-4260.	1.4	79
82	Genetic Modulation of Sickle Cell Disease and Thalassemia. , 2009, , 638-657.		4
83	SPECIAL TOPICS IN HEMOGLOBINOPATHIES. , 2009, , 623-624.		0
84	Population analysis of the alpha hemoglobin stabilizing protein (AHSP) gene identifies sequence variants that alter expression and function. American Journal of Hematology, 2008, 83, 103-108.	4.1	48
85	Association between active genes occurs at nuclear speckles and is modulated by chromatin environment. Journal of Cell Biology, 2008, 182, 1083-1097.	5.2	231
86	Chapter 5 Longâ€Range Regulation of Î±â€Globin Gene Expression. Advances in Genetics, 2008, 61, 143-173.	1.8	30
87	A large deletion in the human Â-globin cluster caused by a replication error is associated with an unexpectedly mild phenotype. Human Molecular Genetics, 2008, 17, 3084-3093.	2.9	26
88	Genetic complexity in sickle cell disease. Proceedings of the National Academy of Sciences of the United States of America, 2008, 105, 11595-11596.	7.1	43
89	Neuronal Death Resulting from Targeted Disruption of the Snf2 Protein ATRX Is Mediated by p53. Journal of Neuroscience, 2008, 28, 12570-12580.	3.6	61
90	A New Dawn for Stem-Cell Therapy. New England Journal of Medicine, 2008, 358, 964-966.	27.0	12

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91	Switching genes on and off in haemopoiesis. Biochemical Society Transactions, 2008, 36, 613-618.	3.4	6
92	The role of the polycomb complex in silencing Î±-globin gene expression in nonerythroid cells. Blood, 2008, 112, 3889-3899.	1.4	51
93	Long-range regulation of ?? globin gene expression during erythropoiesis. Current Opinion in Hematology, 2008, 15, 176-183.	2.5	66
94	Switching Genes On and Off During Hematopoiesis.. Blood, 2008, 112, sci-17-sci-17.	1.4	0
95	Structural consequences of disease-causing mutations in the ATRX-DNMT3-DNMT3L (ADD) domain of the chromatin-associated protein ATRX. Proceedings of the National Academy of Sciences of the United States of America, 2007, 104, 11939-11944.	7.1	138
96	Manipulating the Mouse Genome to Engineer Precise Functional Syntenic ReplacementsÂwith Human Sequence. Cell, 2007, 128, 197-209.	28.9	150
97	Defining the Cause of Skewed X-Chromosome Inactivation in X-Linked Mental Retardation by Use of a Mouse Model. American Journal of Human Genetics, 2007, 80, 1138-1149.	6.2	32
98	Tissue-specific histone modification and transcription factor binding in Î± globin gene expression. Blood, 2007, 110, 4503-4510.	1.4	69
99	Using Genomics to Study How Chromatin Influences Gene Expression. Annual Review of Genomics and Human Genetics, 2007, 8, 299-325.	6.2	33
100	Long-range chromosomal interactions regulate the timing of the transition between poised and active gene expression. EMBO Journal, 2007, 26, 2041-2051.	7.8	224
101	Prevalence of erythrocyte haemoglobin H inclusions in unselected patients with clonal myeloid disorders. British Journal of Haematology, 2007, 139, 439-442.	2.5	12
102	A Regulatory SNP Causes a Human Genetic Disease by Creating a New Transcriptional Promoter. Science, 2006, 312, 1215-1217.	12.6	254
103	A novel deletion causing Î± thalassemia clarifies the importance of the major human alpha globin regulatory element. Blood, 2006, 107, 3811-3812.	1.4	34
104	A novel mutation in the last exon of ATRX in a patient with alpha-thalassemia myelodysplastic syndrome. European Journal of Haematology, 2006, 76, 432-435.	2.2	10
105	Loss of Atrx Affects Trophoblast Development and the Pattern of X-Inactivation in Extraembryonic Tissues. PLoS Genetics, 2006, 2, e58.	3.5	140
106	Coregulated human globin genes are frequently in spatial proximity when active. Journal of Cell Biology, 2006, 172, 177-187.	5.2	192
107	Acquired Î±-thalassemia in association with myelodysplastic syndrome and other hematologic malignancies. Blood, 2005, 105, 443-452.	1.4	95
108	Annotation of cis-regulatory elements by identification, subclassification, and functional assessment of multispecies conserved sequences. Proceedings of the National Academy of Sciences of the United States of America, 2005, 102, 9830-9835.	7.1	133

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109	SW-ARRAY: a dynamic programming solution for the identification of copy-number changes in genomic DNA using array comparative genome hybridization data. Nucleic Acids Research, 2005, 33, 3455-3464.	14.5	87
110	The chromatin-remodeling protein ATRX is critical for neuronal survival during corticogenesis. Journal of Clinical Investigation, 2005, 115, 258-267.	8.2	169
111	The chromatin-remodeling protein ATRX is critical for neuronal survival during corticogenesis. Journal of Clinical Investigation, 2005, 115, 258-267.	8.2	119
112	Acquired somatic ATRX mutations in myelodysplastic syndrome associated with Î± thalassemia (ATMDS) convey a more severe hematologic phenotype than germline ATRX mutations. Blood, 2004, 103, 2019-2026.	1.4	84
113	Ham-Wasserman Lecture. Hematology American Society of Hematology Education Program, 2004, 2004, 1-13.	2.5	25
114	Comparative Analysis of the Â-Like Globin Clusters in Mouse, Rat, and Human Chromosomes Indicates a Mechanism Underlying Breaks in Conserved Synteny. Genome Research, 2004, 14, 623-630.	5.5	29
115	Globin gene activation during haemopoiesis is driven by protein complexes nucleated by GATA-1 and GATA-2. EMBO Journal, 2004, 23, 2841-2852.	7.8	193
116	A conserved truncated isoform of the ATR-X syndrome protein lacking the SWI/SNF-homology domain. Gene, 2004, 326, 23-34.	2.2	53
117	Deletion of the Î±-globin gene cluster as a cause of acquired Î±-thalassemia in myelodysplastic syndrome. Blood, 2004, 103, 1518-1520.	1.4	34
118	Evaluation of alpha hemoglobin stabilizing protein (AHSP) as a genetic modifier in patients with Î² thalassemia. Blood, 2004, 103, 3296-3299.	1.4	102
119	De novo deletion within the telomeric region flanking the human Î± globin locus as a cause of Î± thalassaemia. British Journal of Haematology, 2003, 120, 867-875.	2.5	36
120	Transcription of antisense RNA leading to gene silencing and methylation as a novel cause of human genetic disease. Nature Genetics, 2003, 34, 157-165.	21.4	505
121	Identification of acquired somatic mutations in the gene encoding chromatin-remodeling factor ATRX in the Î±-thalassemia myelodysplasia syndrome (ATMDS). Nature Genetics, 2003, 34, 446-449.	21.4	132
122	Deletion of the mouse Î±-globin regulatory element (HS â^'26) has an unexpectedly mild phenotype. Blood, 2002, 100, 3450-3456.	1.4	53
123	Characterization of a Widely Expressed Gene (LUC7-LIKE; LUC7L) Defining the Centromeric Boundary of the Human Î±-Globin Domain. Genomics, 2001, 71, 307-314.	2.9	31
124	Monosomy for the most telomeric, gene-rich region of the short arm of human chromosome 16 causes minimal phenotypic effects. European Journal of Human Genetics, 2001, 9, 217-225.	2.8	47
125	Expression of Î±- and Î²-globin genes occurs within different nuclear domains in haemopoietic cells. Nature Cell Biology, 2001, 3, 602-606.	10.3	139
126	Comparative genome analysis delimits a chromosomal domain and identifies key regulatory elements in the alpha globin cluster. Human Molecular Genetics, 2001, 10, 371-382.	2.9	151

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127	Sequence, structure and pathology of the fully annotated terminal 2 Mb of the short arm of human chromosome 16. Human Molecular Genetics, 2001, 10, 339-352.	2.9	81
128	Molecular-clinical spectrum of the ATR-X syndrome. American Journal of Medical Genetics Part A, 2000, 97, 204-212.	2.4	208
129	A nonsense mutation of theATRX gene causing mild mental retardation and epilepsy. Annals of Neurology, 2000, 47, 117-121.	5.3	72
130	Mutations in ATRX, encoding a SWI/SNF-like protein, cause diverse changes in the pattern of DNA methylation. Nature Genetics, 2000, 24, 368-371.	21.4	476
131	Î±-Thalassemia resulting from a negative chromosomal position effect. Blood, 2000, 96, 800-807.	1.4	50
132	Î±-Thalassemia resulting from a negative chromosomal position effect. Blood, 2000, 96, 800-807.	1.4	2
133	Comparison of the human and murine ATRX gene identifies highly conserved, functionally important domains. Mammalian Genome, 1998, 9, 400-403.	2.2	64
134	Do LCRs Open Chromatin Domains?. Cell, 1998, 95, 299-302.	28.9	94
135	Human ARHGDIG, a GDP-Dissociation Inhibitor for Rho Proteins: Genomic Structure, Sequence, Expression Analysis, and Mapping to Chromosome 16p13.3. Genomics, 1998, 53, 104-109.	2.9	14
136	The relationship between chromosome structure and function at a human telomeric region. Nature Genetics, 1997, 15, 252-257.	21.4	143
137	Mutations in transcriptional regulator ATRX establish the functional significance of a PHD-like domain. Nature Genetics, 1997, 17, 146-148.	21.4	196
138	The Î±-Thalassemia/Mental Retardation Syndromes. Medicine (United States), 1996, 75, 45-52.	1.0	24
139	X-linked Î±-thalassemia/mental retardation (ATR-X) syndrome: A new kindred with severe genital anomalies and mild hematologic expression. American Journal of Medical Genetics Part A, 1995, 55, 302-306.	2.4	50
140	Mutations in a putative global transcriptional regulator cause X-linked mental retardation with Î±-thalassemia (ATR-X syndrome). Cell, 1995, 80, 837-845.	28.9	583
141	Conservation of Position and Sequence of a Novel, Widely Expressed Gene Containing the Major Human Î±-Globin Regulatory Element. Genomics, 1995, 29, 679-689.	2.9	56
142	The IL-9 Receptor Gene (IL9R): Genomic Structure, Chromosomal Localization in the Pseudoautosomal Region of the Long Arm of the Sex Chromosomes, and Identification of IL9R Pseudogenes at 9qter, 10pter, 16pter, and 18pter. Genomics, 1995, 29, 371-382.	2.9	72
143	Analysis of a 70 kb segment of DNA containing the human Î¶ and Î±-globin genes linked to their regulatory element (HS-40) in transgenic mice. Nucleic Acids Research, 1994, 22, 4139-4147.	14.5	48
144	5 Î±-Thalassaemia. Best Practice and Research: Clinical Haematology, 1993, 6, 117-150.	1.1	94

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145	An unusually large (CA)n repeat in the region of divergence between subtelomeric alleles of human chromosome 16p. Genomics, 1992, 13, 81-88.	2.9	17
146	Stable length polymorphism of up to 260 kb at the tip of the short arm of human chromosome 16. Cell, 1991, 64, 595-606.	28.9	169
147	A truncated human chromosome 16 associated with Î± thalassaemia is stabilized by addition of telomeric repeat (TTAGGG)n. Nature, 1990, 346, 868-871.	27.8	300
148	Structure and expression of the human Î¸l globin gene. Nature, 1988, 331, 94-96.	27.8	53
149	Clinical features and molecular analysis of acquired hemoglobin H disease. American Journal of Medicine, 1983, 75, 181-191.	1.5	49
150	The Interaction of Alpha-Thalassemia and Homozygous Sickle-Cell Disease. New England Journal of Medicine, 1982, 306, 1441-1446.	27.0	305
151	Molecular and Cellular Basis of Hemoglobin Switching. , 0, , 86-100.		3
152	Î± THALASSEMIA. , 0, , 239-240.		6
153	Unusual Types of Î± Thalassemia. , 0, , 296-320.		2
154	Other Sickle Hemoglobinopathies. , 0, , 564-586.		4
155	The Normal Structure and Regulation of Human Globin Gene Clusters. , 0, , 46-61.		8